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22: Furst DE, Breedveld FC, Kalden JR, Smolen JS, Antoni CE, Bijlsma JW, Burmester GR, Cronstein B, Keystone EC, Kavanaugh A, Klareskog L. Related Articles, Links

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☐ **26:** Kuzel TM. Related Articles, Links

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AN   2006187607     MEDLINE
DN   PubMed ID: 16585559
TI   NF-kappaB-inducing kinase is involved in the activation of the
CD28
     responsive element through phosphorylation of c-Rel and
regulation of its
     transactivating activity.
AU   Sanchez-Valdepenas Carmen; Martin Angel G; Ramakrishnan
Parameswaran;
     Wallach David; Fresno Manuel
CS   Centro de Biologia Molecular, Consejo Superior de Investigaciones
     Cientificas, Universidad Autonoma de Madrid, Madrid, Spain.
SO   Journal of immunology (Baltimore, Md. : 1950), (2006 Apr 15)
Vol. 176, No.
     8, pp. 4666-74.
     Journal code: 2985117R. ISSN: 0022-1767.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Abridged Index Medicus Journals; Priority Journals
EM   200605
ED   Entered STN: 5 Apr 2006
     Last Updated on STN: 17 May 2006
     Entered Medline: 16 May 2006
AB   Previous evidence suggested that NF-kappaB-inducing kinase (NIK)
might
     regulate IL-2 synthesis.  However, the molecular mechanism is not
     understood.  In this study, we show that NIK is involved in CD3
plus CD28
     activation of IL-2 transcription.  Splenic T cells from aly/aly
mice (that
     have a defective NIK protein) have a severe impairment in IL-2
and GM-CSF
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but not TNF secretion in response to CD3/CD28. This effect takes place at the transcriptional level as overexpression of alyNIK inhibits IL-2 promoter transcription. NIK activates the CD28 responsive element (CD28RE) of the IL-2 promoter and strongly synergizes with c-Rel in this activity. We found that NIK interacts with the N-terminal domain of c-Rel, mapping this interaction to aa 771-947 of NIK. Moreover, NIK phosphorylates the c-Rel C-terminal transactivation domain (TAD) and induces Gal4-c-Rel-transactivating activity. Anti-CD28 activated Gal4-c-Rel transactivation activity, and this effect was inhibited by a NIK-defective mutant. Deletion studies mapped the region of c-Rel responsive to NIK in aa 456-540. Mutation of several serines, including Ser471, in the TAD of c-Rel abrogated the NIK-enhancing activity of its transactivating activity. Interestingly, a Jurkat mutant cell line that expresses one of the mutations of c-Rel (Ser471Asn) has a severe defect in IL-2 and CD28RE-dependent transcription in response to CD3/CD28 or to NIK. Our results support that NIK may be controlling CD28RE-dependent transcription and T cell activation by modulating c-Rel phosphorylation of the TAD. This leads to more efficient transactivation of genes which are dependent on CD28RE sites where c-Rel binds such as the IL-2 promoter.

L2 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 2
AN 2001370772 MEDLINE
DN PubMed ID: 11278268
TI Effects of the NIK aly mutation on NF-kappaB activation by the Epstein-Barr virus latent infection membrane protein, lymphotoxin beta receptor, and CD40.
AU Luftig M A; Cahir-McFarland E; Mosialos G; Kieff E
CS Departments of Microbiology and Molecular Genetics and Medicine, Program in Virology, Harvard Medical School, Boston, Massachusetts 02115, USA.
NC CA47006 (NCI)
SO The Journal of biological chemistry, (2001 May 4) Vol. 276, No. 18, pp.

14602-6. Electronic Publication: 2001-03-14.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200106
ED Entered STN: 2 Jul 2001
Last Updated on STN: 5 Jan 2003
Entered Medline: 28 Jun 2001
AB Homozygosity for the aly point mutation in NF-kappaB-inducing kinase (NIK)
results in alymphoplasia in mice, a phenotype similar to that of homozygosity for deletion of the lymphotoxin beta receptor (LTbetaR). We now find that NF-kappaB activation by Epstein-Barr virus latent membrane protein 1 (LMP1) or by an LMP1 transmembrane domain chimera with the LTbetaR signaling domain in human embryonic kidney 293 cells is selectively inhibited by a wild type dominant negative NIK comprised of amino acids 624-947 (DN-NIK) and not by aly DN-NIK. In contrast, LMP1/CD40 is inhibited by both wild type (wt) and aly DN-NIK. LMP1, an LMP1 transmembrane domain chimera with the LTbetaR signaling domain, and LMP1/CD40 activate NF-kappaB in wt or aly murine embryo fibroblasts. Although wt and aly NIK do not differ in their in vitro binding to tumor necrosis factor receptor-associated factor 1, 2, 3, or 6 or in their in vivo association with tumor necrosis factor receptor-associated factor 2 and differ marginally in their very poor binding to IkappaB kinase beta (IKKbeta), only wt NIK is able to bind to IKKalpha. These data are compatible with a model in which activation of NF-kappaB by LMP1 and LTbetaR is mediated by an interaction of NIK or a NIK-like kinase with IKKalpha that is abrogated by the aly mutation. On the other hand, CD40 mediates NF-kappaB activation through a kinase that interacts with a different component of the IKK complex.